Dengue

and Mosquito Control

on Rarotonga

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Contents

The disease and the mosquitoes

Dengue is a severe flu-like illness of the tropics. It is caused by a virus, and spread by the day-biting Aedes *mosquitoes. There have been seven outbreaks or epidemics on Rarotonga since 1976.*

Dengue disease and viruses

Dengue symptoms are very high fever, and two of: a severe frontal headache, painful joints and muscles, or a rash. The fever stage usually lasts 5-7 days (limits 3-14), and after a week or two of convalescence most people are fully recovered, although some experience weakness and tiredness for several weeks. Clinically, the fever stage is associated with a decrease of blood platelets, which are the plate-like cells that clot blood.

Dengue is a severe illness, but in some cases after 3-5 days of fever the patient further deteriorates instead of improving, and can die in the absence of adequate medical care. When this happens the disease is called Dengue Haemorrhagic Fever (a.k.a. Haemorrhagic Dengue). Clinically the early hallmark feature of Haemorrhagic Dengue is increased plasma leakage, usually associated with a low platelet count (a.k.a. thrombocytopaenia) and signs of haemorrhage bleeding. In recognition that Dengue Fever (DF) and Dengue Haemorrhagic Fever (DHF) are a continuum of the same disease, and that haemorrhage is not the early hallmark of the latter, WHO and others are now using Dengue and Severe Dengue to name the two forms of dengue.

Although scary, Severe Dengue is easily detected by a doctor and easily treated at a hospital, and the death of patients under care is very rare.

Dengue was an uncommon illness until it became widespread in Southeast Asia in the 1950s, and at that time the first cases of Severe Dengue were also reported. Both Dengue and Severe Dengue have become more widespread and frequent in the last 50 years. WHO estimates that 50 million people get dengue each year, including around half-a-million with Severe Dengue, which is relatively more common among young people. Without specialised treatment 20% of Severe Dengue patients die, but with suitable treatment the mortality is usually less than one-per-1000 (i.e. 0.1%).

Dengue is caused by a virus, which means there is no medicine to kill it when it infects a person; unlike bacterial infections, which can be treated with antibiotics (=bacteriocides). Treatment is simply supportive: plenty of fluids to prevent dehydration, lowering the temperature, and reducing the pain with paracetamol (NOT aspirin, which can promote bleeding). And, of course, ask your doctor about monitoring for signs of Severe Dengue, which will require hospitalisation and transfusions.

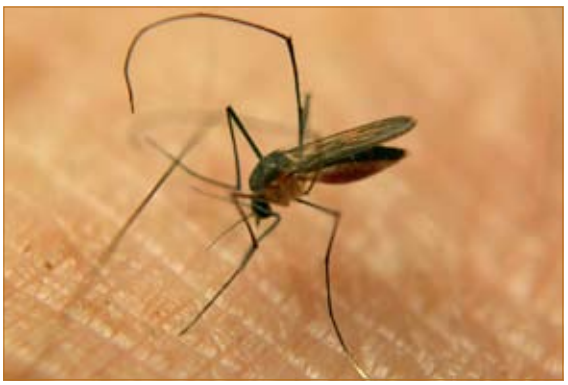
There are four serotypes of the dengue virus (DEN-1, DEN-2, DEN-3 & DEN-4) and after being infected with one serotype you have a life-time immunity to that serotype, but not the other three serotypes. Any of the four serotypes of dengue can be present in a severe outbreak, and it is not known why in some outbreaks a few individuals develop the severe form of dengue. There is strong support for the hypothesis that a secondary infection by a different serotype of dengue is more likely to be Severe, because of antibody enhancement of viral activity. This hypothesis also predicts that Severe Dengue is more likely if an outbreak is within five years of a previous outbreak. This prediction was supported by data from French Polynesia on the last ten outbreaks. Of the five that were within five years of a previous outbreak, three had severe cases and two did not; and of the five outbreaks with more than five years to the previous outbreak, none had severe cases.

When you think you have dengue, it is important to consult a doctor so that the progress of your dengue is being competently monitored. If you have had dengue in a previous outbreak, the “antibody dependent enhancement” (ADE) hypothesis indicates that in a new outbreak you should take extra precautions to not become infected. The alternative hypothesis, the “viral virulence” hypothesis, which predicts that some serotypes are more virulent, continued to have support in Brazil in 2002 when primary infections were associated with Severe Dengue in a DEN-3 outbreak. It is now known that each serotype has genetic variations forming distinctive genotypes, and some genotypes have been more closely linked to Severe Dengue. Life-time personal immunity after being infected with a particular serotype is to that serotype and all its genotypes.

A vaccine is a medicine that gives immunity to a particular viral disease by pre-activating the antibody system. A vaccine for dengue is difficult to develop because it needs to activate different antibodies for each of the four dengue serotypes at the same time. If the vaccine was for only one serotype, the “antibody dependent enhancement” hypothesis predicts that the vaccine would be a risk factor for Severe Dengue if a person became infected with another serotype.

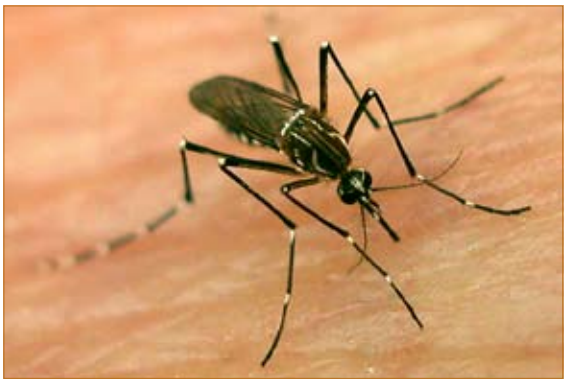
Rarotonga mosquitoes

Rarotonga has four species of mosquito. The two dull grey-brown, night-biting mosquitoes are *Culex* species and they do not transmit the dengue virus, nor any other disease in the Cook Islands. They breed in large and small pools of water, as in the swamplands.



Culex quinquefasciatus

Aedes polynesiensis



Aedes aegypti

The two black-and-white stripped mosquitoes that bite during the day are *Aedes* species, *Aedes polynesiensis* and *Aedes aegypti*, and they both transmit dengue from one person to another. Dengue is not transmitted from other mammals, such as cats and dogs, or from birds - only person-to-person. Vertical transmission of the virus from the mosquito to her offspring has been known to occur, but it is rarely significant in maintaining the dengue virus.

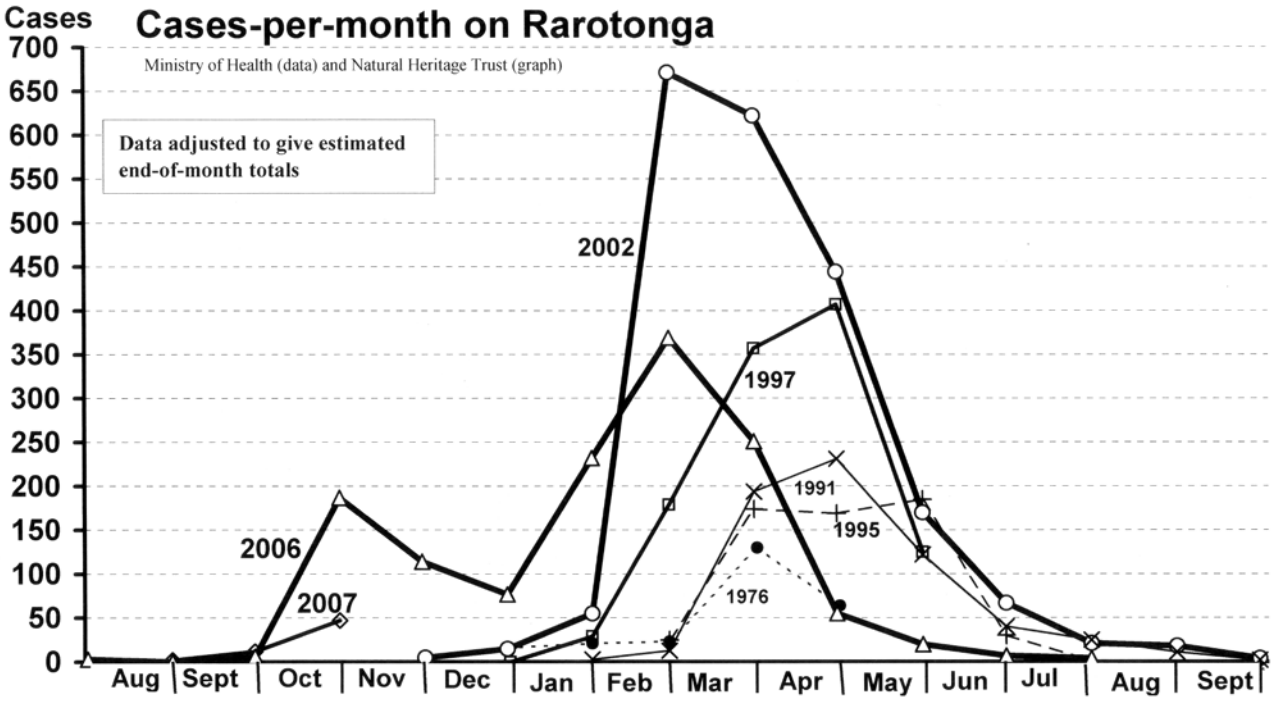
The two *Aedes* mosquitoes lay their eggs and grow in small containers of water, in either artificial ones like empty tins and old tyres, or natural ones like holes in trees and opened coconuts. Both *Aedes* use containers around dwellings, but *Aedes polynesiensis* also breeds in holes in trees and small natural containers throughout the mountains and on the motu/islets. *Aedes aegypti* is a domestic mosquito - it breeds only near dwellings and commonly rests inside dwellings.

Female mosquitoes need a sip of blood to develop each batch of eggs. After a blood meal they lay dozens of eggs on the water surface or just above the water line. The larva (a.k.a. wrigglers) feed mainly on bacteria and micro-algae, and new adults can emerge 10-12 days after the eggs were laid. The eggs can become dormant if the water dries up and re-commence development when the water returns. Although the two *Aedes* tend to prefer slightly stagnant water, *Aedes aegypti* is also happy in drinking-quality water.

Of the two *Aedes*, *Aedes aegypti* is the most harmful. This is because it lives exclusively near human habitation, often resting inside dwellings, and feeds almost exclusively on human blood, which it does frequently (once every 2-3 days).

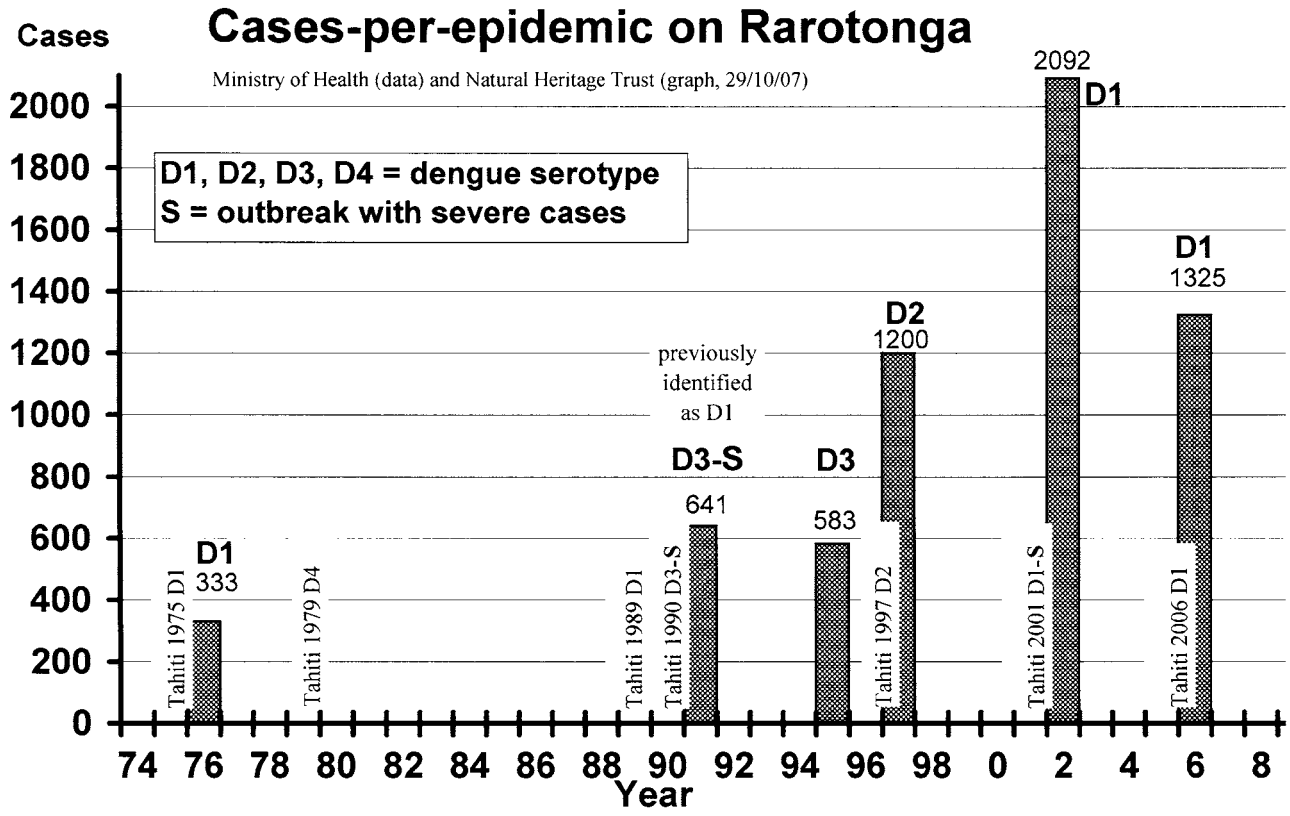
Rarotonga dengue epidemics

The first significant dengue outbreak on Rarotonga was in 1976. While 333 people with dengue consulted doctors, it was estimated that around 5,000 of the 9,000 residents had dengue, although often the symptoms were mild or absent.



Graphs are presented for the seven dengue epidemics on Rarotonga to 2006. In each case some outer islands also had outbreaks but here we analyse the situation on Rarotonga. The Cases-per-month graph shows that the first three epidemics accelerated in March; the 1997 and 2002 accelerated in February; and the 2006 outbreak was very different in that it accelerated in October then dropped back in November and December before accelerating again in January. With the exception of the 2006 outbreak which had two peaks, the rest peaked for three months and were then finished. The two factors involved in ending an outbreak are the rising immunity of the population and the density of dengue-transmitting *Aedes* mosquitoes.

There is a theory that an outbreak will stop, even when *Aedes* are abundant, when 80% of the population is immune (a.k.a. 80% “herd immunity”). This theory was supported in the Society Islands 2001 DEN-1 outbreak when it was estimated that 56% were immune from the 1989 DEN-1 outbreak and a further 23% were infected in 2001 before the outbreak ended. Rarotonga had DEN-1 in 2002 and again in 2006, with 2092 and 1325 reported-cases respectively. The 2006 outbreak shows that the 2002 outbreak did not stop because of 80% “herd immunity”, and we can conclude that it stopped when the *Aedes* decreased below the density required to maintain the outbreak, because of lower humidity and cooler temperatures.



The Cases-per-epidemic shows that more cases were reported in the last three outbreaks and while this might indicate that relatively more people were infected, it might also indicate that more people were being cautious and were consulting medical staff. Only the 1991 outbreak had Severe Cases and there were no deaths after platelet transfusions commenced. The graph also shows that for six of the seven Rarotonga epidemics Tahiti had an outbreak of the same serotype immediately prior to the Rarotonga outbreak, and in most cases the Rarotonga outbreak was clearly associated to travellers from Tahiti. The exception was the 1995 outbreak, which was associated with travellers from Fiji.

Is dengue endemic (constantly present) on Rarotonga? In French Polynesia it was found that after the 1971 DEN-2, 1975 DEN-1, 1979 DEN-4, 1989 DEN-1 epidemics that the serotype persisted at a very low level of infection (~3%) until a couple of months into the next epidemic by a different serotype (introduced from abroad) - the extreme case was the persistence of DEN-4 from 1979 for ten years. When a serotype of dengue persists at low levels after an epidemic it is said to be "endemic". For a serotype to become endemic it needs a large population, so there will still be many who did not become immune during the epidemic. In French Polynesia, the 2006 DEN-1 outbreak following the 2001 DEN-1 outbreak, was the first time they had the same serotype in back-to-back outbreaks, and although they know that the DEN-1 virus of 1989 disappeared during the DEN-3 outbreak of 1990, it is most likely that the DEN-1 that arrived in 2002 persisted as an endemic and caused the 2006 outbreak.

Rarotonga also had back-to-back DEN-1 in 2002 and 2006. The Ministry of Health traced the second outbreak to travellers from Tahiti, so it seems that the virus of 2002 did not persist for the three years as an endemic. Regardless of the origin of the virus, it is obvious that a high density of *Aedes* coupled with a relatively low "herd immunity" was sufficient to launch the 2006 epidemic. As to the increase of likely-dengue cases in September and October 2007, it will be interesting to see if the DEN-1 of 2006 has been a low-lying endemic for a few months before an increase of mosquito activity enabled it to launch a new endemic or if a traveller introduced a different serotype.

Countries with very large urban populations in tropical Asia and South America often have an endemic serotype, and in several cases they have two or three serotypes co-circulating (known as hyper-endemicity), and this is thought to be a risk factor for Severe Dengue. Although French Polynesia usually had two serotypes circulating at the beginning of a new outbreak, the co-circulation has never persisted.

The origin of a typical epidemic

Tahiti typically gets a new outbreak when a traveller brings a suitable serotype from abroad. Rarotonga typically gets a new outbreak when a traveller brings a suitable serotype from abroad - usually from Tahiti!

When a person is infected by a mosquito, the viruses reproduce in him/her without causing illness for 3 to 14 days (usually 4-7) before they have enough viruses to cause the fever and other symptoms. This means that any healthy traveller can arrive and unknowingly feed dengue viruses to the female mosquitoes that take his/her blood. The viruses take about 7 days (8-12) to develop in the mosquito's salivary glands and then she is very infective to everyone she bites for her life-time of around a month (2-12 weeks). During her life-time she usually hangs around the same area if there are small containers with water for egg-laying and people to provide more blood, or she might gradually move to another areas up to a couple of hundred metres away.

When a traveller arrives with a dengue virus there are two scenarios. The best scenario is that most people already have immunity and only a few people are infected. The worst scenario is that most people lack immunity and if mosquitoes are abundant a full epidemic is launched.

Managing dengue-spreading mosquitoes

Dengue, without even considering Severe Dengue, is a very stressful and debilitating illness. Scientists are working on new and better ways to control the mosquitoes that spread it, and on affordable vaccines to give immunity. Unfortunately, these are at best a few years away, and in the meantime another summer - another mosquito heaven - is approaching. We do not want to stop travellers, and we cannot identify those who arrive with the pre-fever stage of dengue. What can we do?

Aedes larvae control

Aedes mosquitoes increase dramatically in summer with the warmer, wetter and more humid conditions. The most socially and environmentally friendly way to reduce their density is to destroy all breeding sites around houses and where people congregate. This means that it is up to everyone in the community to take responsibility and ensure that their area is free of *Aedes* breeding sites - all not-required small containers that can hold water should be removed or filled in, and all required containers should be covered with a lid or mosquito cloth, or the water replaced every few days. Although stagnant water is preferred for egg-laying, fresh water is quite acceptable to *Aedes aegypti*.

The most effective method of reducing the *Aedes* population is to support the Public Health Tutaka and remove or destroy every small water-container in and around your home. Because *Aedes* can fly around 200 metres in search of blood, your neighbours need to destroy breeding sites also.

The Ministry of Health has also been experimenting with a larvicidal bacteria known as Bti (*Bacillus thuringiensis israelensis*) in the form of Vectobac for six years. It has the advantage of being lethal to mosquito larvae, but harmless to people, birds and fish because it passes through the gut without being absorbed. It has the disadvantage of needing to be re-applied every week or so, because it is destroyed by sunlight. This larvicide is ideal for use against the larvae of *Culex* mosquitoes because they develop in large water containers, such as the puddles and pools in the swamplands. It is obviously beneficial to reduce the night-biting mosquitoes because they are a serious nuisance, but they do not spread dengue. The bacteria would be useful against *Aedes* larvae in larger domestic water-containers that cannot be removed or covered, but it would be counterproductive to use it on the countless small containers that ought to be removed or filled-in or frequently emptied. Support the Tutaka! Monomolecular layers of oils can also be used to suffocate mosquito larvae.

Aedes adult control

If larval control is not 100% then there will always be some adult *Aedes* to carry the virus from one person to another, and even at low densities they can effectively spread the virus when the “herd immunity” for the particular dengue serotype is low (less than 40%). The reaction to the first few cases of dengue in most Pacific island countries has been to isolate the person from mosquitoes with repellents, mosquito nets, and suitable clothing; to do perifocal misting or fogging with an insecticide to kill all *Aedes* that

may have already taken a blood-meal; and to do perifocal destruction of *Aedes* breeding sites. Sometimes during an outbreak there has also been mass misting or fogging with insecticides, usually from trucks driving along roads. The reason for Ultra Low Volume (ULV) spraying, or Thermal Fogging, is to release extremely small droplets of insecticide into the air where they remain suspended for a considerable time to kill mosquitoes that come into contact with them.

The question is: are the insecticides harmful to people and/or the environment? By harmful, we mean are they acutely toxic to people; or in the longer term, do they cause cancer, birth defects, or other illnesses in people? Do they damage the environment by killing birds, fish, or other animals, including useful insects?

Reslin

For the mass control of mosquitoes by fogging or ULV spraying, the most widely used insecticides are the organophosphate Malathion, or one of several pyrethroids. The Ministry of Health has used Malathion, but is presently using Reslin®, which contains 50g/L of the pyrethroid Bioresmethrin, 400g/L of Piperonyl Butoxide (PBO), and 370g/L of hydrocarbon liquid as a solvent. PBO is an enhancer or synergist that makes the insecticide more toxic to insects by preventing the insect from neutralising it. The recommended application rate on the label is up to 100ml of premix-Reslin per hectare (10,000m²) or 5g Bioresmethrin/ha. Another Reslin formulation, widely used in the USA is Aqua-Reslin®, which consists of 200g/L the pyrethroid Permethrin, 200g/L PBO, and inert ingredients including petroleum distillate; and the recommended application rate on the label is up 40ml premix per hectare or 8g Permethrin/ha. In the following analysis I will often refer to Permethrin rather than Bioresmethrin because the two chemicals are very similar, but Permethrin is more widely used in the USA and has been studied more extensively. Both were first registered for use in the USA in the 1970s.

What are pyrethroids?

Pyrethrins are natural insecticidal chemicals extracted from the flowers of a Chrysanthemum, and the natural mixture of the six Pyrethrins is known as Pyrethrum. Pyrethroids are synthetic or man-made chemicals that have the insecticidal features of the Pyrethrins, but they are more potent and remain potent for a longer time. There are many different pyrethroids used as insecticides, and their names end with “-thrin”.

Almost all the Raid, Mortein, PeaBeu, Budget and Ultrapel aerosols or coils sold in Rarotonga supermarkets and shops contain synthetic pyrethroids, such as: Tetramethrin, Permethrin, Allethrin, Cypermethrin, and Bioresmethrin. The one aerosol with natural Pyrethrins has 88% synthetic pyrethroids and only 12% natural Pyrethrins as insecticidal chemicals - and lots of PBO enhancer (see Appendix). In addition to their domestic use, synthetic pyrethroids are used on all aircraft to New Zealand and Australia. The residual disinsection is 2% Permethrin every eight weeks to give a layer 0.2g/m² Permethrin on all internal surfaces. The alternative is aerosol spraying of the cabin with the passengers seated using 2% d-Phenothrin at 10g aerosol per 28m³. Synthetic pyrethroids have been in use for over 30 years and account for 25% of the world insecticide market, and they are used against a wide variety of pest insects.

Synthetic Pyrethroids and the natural Pyrethrins are powerful neurotoxins to insects, fish and crustaceans, but are very mild toxins to mammals and birds. However, beware, all mammals are not equal; for example, pyrethroid flea compounds (correctly used) are harmless to dogs, but are very toxic to cats.

Acute toxicity to people

The basic principle of toxicity is: “Nothing is poison and everything is poison; the difference is in the dose.” This is based on a statement made 500 years ago by Paracelsus, the father of modern toxicology. Therefore, the real question is not “is this chemical poisonous”, it is “at what dose is this chemical poisonous”.

To measure the lethal dose of chemicals scientists do not test them on people, but use animals, especially rats, as proxies or surrogates. In the experiments they feed the rats the chemical until they find the single dose that kills 50% of them, and that amount is known as the oral LD50 or oral Median Lethal Dose - as mg/kg(body weight). The oral LD50 is a measure of the short-term Toxicity or Acute Toxicity of a chemical taken through the mouth.

The oral LD50s for some chemicals are:

- natural Nicotine 5mg/kg
- natural Caffeine (as in coffee and tea) 200mg/kg
- synthetic Malathion 1,000mg/kg
- synthetic Permethrin 1,000mg/kg
- natural Pyrethrins 5,000mg/kg
- synthetic PBO 5,000mg/kg
- synthetic Bioresmethrin 5,000mg/kg
- synthetic Tetramethrin 5,000mg/kg

The chemicals above are ranked from most toxic (small LD50) to least toxic, and it is immediately apparent that being a natural or synthetic chemical does not help us to predict its toxicity. A further consideration is that a chemical might be more or less toxic for different genders or age groups of rats. For example, young rats are three times more susceptible to Permethrin poisoning, and we would therefore conclude that young people would be much more susceptible than adults.

A less technical statement of toxicity is that a fatal dose of caffeine for a 80kg person is about 16g - the amount in ten litres of espresso coffee (100mg caffeine/60ml cup). Undiluted Reslin is about ten times more lethal than espresso coffee and a fatal dose would be about one litre, i.e. the amount used to spray more than ten hectares of land for mosquito control.

Toxicity to other animals

Permethrin and Malathion have low toxicity to birds, although birds are around twice as susceptible as mammals. In contrast, fish are extremely susceptible to poisoning by Permethrin and Malathion (0.1 and 0.05 parts per million respectively, kills 50%), and aquatic invertebrates are also extremely susceptible to poisoning.

Absorbed Permethrin and Malathion do not accumulate in the body nor in the food chain. Un-absorbed Permethrin and Malathion are ranked as breaking down rapidly in

the environment. The time chemicals take to breakdown in the environment is measured as a Half-life, which is the time after which 50% remains, and after five Half-lives 3% will remain. The half-lives for Permethrin are: in soil 30 days; on leaves 10 days; in sunlight 5 days; and in water 3 days. Malathion half-lives are 30 days in soil and 5 days in water.

Carcinogenicity to humans

Carcinogenicity is a measure of the likelihood of prolonged exposure to a chemical causing cancer or other abnormal cell growth, such as benign tumours. In more than 25 years of use and evaluation there is no direct evidence that Permethrin causes cancer in people. However there are repeatable experiments that show that mice fed more than 300mg/kg(body weight)/day for 100 days develop benign tumours, and similar experiments with rats gave inconclusive results. In less technical terms this means that an 80kg adult drinking 500ml/day of undiluted Reslin for 100 days would probably develop some benign tumours.

Even though the dose to cause a benign tumour is very high, the US Environmental Protection Agency (EPA) states that Permethrin is “Likely to be Carcinogenic to Humans by the oral route. This classification was based on two reproducible benign tumour types (lung and liver) in the mouse, ambiguous evidence of carcinogenicity in Long-Evans rats, and supporting structural activity relationship information.” Natural Pyrethrins have caused benign tumours in the thyroid and liver of rats, and therefore like Permethrin they are also likely to be carcinogenic to people.

Is Reslin safe to use as a ULV spray?

Aqua-Reslin and to a lesser extent Reslin are both lethal toxins (at very high single doses) and both probably cause benign tumours (at high-level prolonged doses). The question is, at what dose are they effective against mosquitoes but harmless to people and the environment?

It is very difficult for an individual, be they science-trained or not, to make such judgements even with the abundance of information on the Internet. The approach used here is to look at the judgements made by responsible and accountable groups in developed countries. In this case, I am using the evaluation by the United States Environmental Protection Agency (EPA), and the evaluation by the Health Board of Westchester County in New York.

The EPA is responsible for registering chemicals that can be used in the USA, and for detailing the conditions of use. Permethrin was first registered for use by the EPA in 1979, and because of advances in science and the development of more stringent standards it was re-evaluated in 2006. After re-evaluating its ability to cause cancer, ability to cause birth defects, and its toxicity to people and other animals, such as fish, the EPA re-registered it as a safe insecticide when used according to the label for UVL spraying to control adult mosquitoes. To protect fish and other aquatic animals the EPA prohibited ULV spraying within 450 feet (140 metres) of ponds, lakes and waterways.

A very detailed evaluation of spraying for mosquitoes with Permethrin in Aqua-Reslin (and also for Malathion) was undertaken in 2001 by the Health Board of Westchester Country New York. They evaluated the cancer and non-cancer risks to people, using a

worst case scenario of ULV spraying children four times-a-season from a distance of 8 metres. While noting the likelihood of short-term eye, nose and respiratory effects, they concluded that there were no projected cancer or non-cancer health issues. They recommended that the public be informed before spraying so they could avoid the spray, if they wished. The Board evaluated risks to other animals, including fish and aquatic invertebrates. They concluded that at mosquito-control levels, the threat to fish and invertebrates near the surface of ponds was negligible, and that the risk could be further reduced by reducing the drift reaching ponds, and by reducing surface absorption by using droplets of less than 30µm (micrometers). To reduce impacts on bees, they concluded that spraying should be avoided from two hours after dawn to two hours before dusk.

At present, the ULV spray mix used by Public Health is 500ml Reslin per 20L water, which is 25g of Bioresmethrin per 20L. Using the recommended application rate on the label, which is in line with the EPA 2006 Permethrin recommendations, the 20L should cover at least five hectares, and the droplet size should be less than 30µm. This means that a 100m radius perifocus (i.e. three hectares) should use less than 10L of the present Public Health mix.

Conclusion

If everyone destroyed *Aedes* breeding sites, the chance of a dengue epidemic would be reduced - **support the Tutaka**!

Nevertheless there will always be sporadic cases of dengue, and given the right conditions they can launch an epidemic, which will typically last three months and cause serious illness for over a thousand people. Unfortunately, by the time each case is detected, it is most likely that mosquitoes have already sucked up the virus and will soon start spreading it to other people. To reduce the likelihood of an epidemic, immediate and intense action is required in a 200m-perifocus (i.e. 200m radius):

- increased effort to destroy *Aedes* breeding sites
- protection against being bitten by mosquitoes with repellents, light clothes and mosquito nets
- increasing the domestic use of pyrethroid and Pyrethrin aerosol sprays and mosquito coils; and where possible, increasing the use of other systems to attract and destroy mosquitoes.

In the 100m-perifocal area, household leaders should be consulted before ULV spraying or fogging. If ULV spraying is undertaken, people should be advised in advance so they can remove themselves from the area, if they wish. If spraying is not undertaken because the consensus is against it, or because there are organic farms or pond fisheries within or adjacent to the 100m-perifocal, then efforts should be doubled to implement the 200m-perifocal procedures for personal and household protection - and this should be monitored.

When there are about 20 cases-a-week and they are in dispersed locations, the virus has won and an epidemic is under way. It will typically run for 3-4 months and die out when our herd immunity is around 80%, or lower humidity and cooler temperatures

significantly reduce the mosquito population. During an epidemic people should destroy all *Aedes* breeding sites in their neighbourhood and reduce the likelihood of being bitten by female *Aedes* mosquitoes at home and at work. Mass spraying from roads is a serious imposition on people and there is no conclusive evidence that its reduces or shortens an epidemic.

Useful resources

WHO factsheet (2002) Dengue and dengue haemorrhagic fever:
http://www.who.int/mediacentre/factsheets/fs117/en/

Analysis of the 2001 DEN-1 epidemic in French Polynesia with Severe Dengue:
http://www.spc.int/phs/PPHSN/Outbreak/Reports/Dengue_report2001-FrenchPolynesia.pdf

Analysis Dengue in French Polynesia to 1997:
http://archives.hellis.org/documents/dbull/dbv22p74.pdf

EPA Overview of Permethrin Risk Assessment 2005:
http://www.up3project.org/documents/Permethrin_PRA_Overview.pdf

The EPA 2006 re-registration of Permethrin outlining usage:
http://www.epa.gov/oppsrrd1/REDs/factsheets/permethrin_fs.htm

Health Board of Westchester County evaluation of insecticides (2001):
http://www.westchestergov.com/hdbooklets/StingEIS/DGEISfiles/Executive%20Summary.pdf

Appendix: Domestic insecticides available in Rarotonga

Raid 350g Multipurpose	4.0g/kg Tetramethrin	1.0g/kg Permethrin	
Raid 350g Fly & Insect Killer	3.5g/kg Tetramethrin	1.0 g/kg Permethrin	1.0g/kg Allethrin
Mortein 200g Fast knockdown	3.8g/kg Tetramethrin	1.2g/kg Bioallethrin	0.8g/kg Bioresmethrin
Mortein 250g Surface Spray	2.0g/kg Cypermethrin	0.7g/kg Imiprothrin	
PeaBeu Fast Kill	2.1g/kg Bioallethrin	0.4g/kg Bioresmethrin	
PeaBeu Surface	2.8g/kg Permethrin	1.4g/kg Tetramethrin	
Budget Fly Spray	3.0g/kg Tetramethrin	15g/kg PBO	
Ultrapel 305g	4.4g/kg Allethrin 3.5g/kg Tetramethrin	1.1g/kg Pyrethrins 45g/kg PBO	
Mortein DIY Bomb 3 x 125g	10.0g/kg Permethrin	0.8g/kg Fenoxycarb	
Mortein, Mos. coils 10, total 120g	2.0g/kg Allethrin		
Fish A, Mos. coils	2.0g/kg d-Allethrin		
Raid Portable Repeller	220.0g/kg Transfluthrin		